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NEWS 8 APR 30 GENBANK reloaded and enhanced with Genome Project ID field
NEWS 9 APR 30 CHEMCATS enhanced with 1.2 million new records
NEWS 10 APR 30 CA/CAplus enhanced with 1870-1889 U.S. patent records
NEWS 11 APR 30 INPADOC replaced by INPADOCDB on STN
NEWS 12 MAY 01 New CAS web site launched
NEWS 13 MAY 08 CA/CAplus Indian patent publication number format defined
NEWS 14 MAY 14 RDISCLOSURE on STN Easy enhanced with new search and display fields
NEWS 15 MAY 21 BIOSIS reloaded and enhanced with archival data
NEWS 16 MAY 21 TOXCENTER enhanced with BIOSIS reload
NEWS 17 MAY 21 CA/CAplus enhanced with additional kind codes for German patents
NEWS 18 MAY 22 CA/CAplus enhanced with IPC reclassification in Japanese patents
NEWS 19 JUN 27 CA/CAplus enhanced with pre-1967 CAS Registry Numbers
NEWS 20 JUN 29 STN Viewer now available
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NEWS 24 JUL 02 SCISEARCH enhanced with complete author names
NEWS 25 JUL 02 CHEMCATS accession numbers revised
NEWS 26 JUL 02 CA/CAplus enhanced with utility model patents from China
NEWS 27 JUL 16 CAplus enhanced with French and German abstracts
NEWS 28 JUL 18 CA/CAplus patent coverage enhanced

NEWS EXPRESS 29 JUNE 2007: CURRENT WINDOWS VERSION IS V8.2,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 05 JULY 2007.

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L2 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2001:759961 CAPLUS
DOCUMENT NUMBER: 136:161034
TITLE: Anti-TNF- α Properties of New 9-Benzyladenine Derivatives with Selective Phosphodiesterase-4-Inhibiting Properties
AUTHOR(S): Reimund, Jean-Marie; Raboisson, Pierre; Pinna, Guillaume; Lugnier, Claire; Bourguignon, Jean-Jacques; Muller, Christian D.
CORPORATE SOURCE: Laboratoire de Pharmacologie et Physico-Chimie des Interactions Cellulaires et Moleculaires, UMR 7034 du CNRS, Universite Louis Pasteur de Strasbourg, UFR de Sciences Pharmaceutiques, Illkirch, 67401, Fr.
SOURCE: Biochemical and Biophysical Research Communications (2001), 288(2), 427-434
CODEN: BBRCA9; ISSN: 0006-291X

PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In inflammatory cells, intracellular cAMP concentration is regulated by cyclic nucleotide phosphodiesterases 4. Therefore, PDE4 inhibition appears as a rational goal for treating acute or chronic inflammatory diseases. Selective PDE4 inhibitors have been developed, but due to unwanted side effects, search for new selective PDE4-inhibitors had to be pursued. Recently, Boichot et al. (J. Pharmacol. Exp. Ther. (2000) 292, 647-653) showed that 9-benzyladenine derivs. are selective PDE4 inhibitors. In vivo data in animals suggested that they may induce fewer side effects (emesis). We examined the effects of new 9-benzyladenines on TNF- α , interleukin (IL)-1 β , IL-6 and IL-8 production by lipopolysaccharide-activated peripheral blood mononuclear cells, and compared them to other PDEs inhibitors. Selected potent 9-benzyladenines, strongly inhibited TNF- α production. Interleukin-1 β , IL-6, and IL-8 production was not significantly affected. Our results suggest that some of these new adenines (i.e., NCS 675 and NCS 700), may be potential therapeutic candidates for the treatment of inflammatory diseases. (c)
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L2 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN.
ACCESSION NUMBER: 2001:670031 CAPLUS
DOCUMENT NUMBER: 136:31471
TITLE: Urodilatin, a natriuretic peptide stimulating particulate guanylate cyclase, and the phosphodiesterase 5 inhibitor dipyridamole attenuate experimental pulmonary hypertension. Synergism upon coapplication
AUTHOR(S): Schermuly, Ralph Theo; Weissmann, Norbert; Enke, Beate; Ghofrani, Hossein Ardeschir; Forssmann, Wolf Georg; Grimminger, Friedrich; Seeger, Werner; Walmrath, Dieter
CORPORATE SOURCE: Department of Internal Medicine, Justus-Liebig-University Giessen, Giessen, D-35392, Germany
SOURCE: American Journal of Respiratory Cell and Molecular Biology (2001), 25(2), 219-225
CODEN: AJRBEL; ISSN: 1044-1549
PUBLISHER: American Thoracic Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In a model of acute pulmonary hypertension in intact rabbits, the authors investigated the vasodilatory potency of intravascularly administered urodilatin, a renal natriuretic peptide type A known to stimulate particulate guanylate cyclase. Urodilatin infusion was performed in the absence and presence of the phosphodiesterase (PDE) type 5 inhibitor dipyridamole. Stable pulmonary hypertension was evoked by continuous infusion of the thromboxane mimetic U46619, resulting in approx. doubling of the pulmonary artery pressure (PAP). When infused as sole agents, both urodilatin and dipyridamole dose-dependently attenuated the pulmonary hypertension, with doses for a 20% decrease in PAP being 30 ng/kg min for urodilatin and 10 μ g/kg min for dipyridamole. A corresponding decrease in systemic arterial pressure (SAP) was noted to occur in response to both agents. Sequential i.v. administration of a subthreshold dose of dipyridamole (1 μ g/kg min), which per se did not affect pulmonary and systemic hemodynamics, and a standard dose of urodilatin (30 ng/kg min) resulted in a significant amplification of both the PAP and the SAP decrease in response to the natriuretic peptide. At the same time, manifold enhanced plasmatic cyclic guanosine monophosphate (cGMP) levels were detected. Aerosolized dipyridamole also dose-dependently attenuated pulmonary hypertension, with only 1 μ g/kg min being sufficient for a

20% decrease in PAP, with no SAP decline. Preceding administration of subthreshold aerosolized dipyridamole (50 ng/kg min) did, however, cause only a minor amplification of the pulmonary vasodilatory response to a subsequently infused standard dose of urodilatin. In conclusion, this is the 1st study to show that urodilatin does possess vasodilatory potency in the pulmonary circulation, and enhanced blood plasma levels of cGMP and synergy with the PDES inhibitor dipyridamole both strongly suggest that this effect proceeds via guanylate cyclase activation. The effect of infused urodilatin is, however, not selective for the pulmonary vasculature, as the systemic vascular resistance declines in a corresponding fashion.

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